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Comment on Giancola (2000)**

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## **Cognition, Emotion, and the Alcohol-Aggression Relationship**

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## Abstract

Dr. Giancola's thesis that the alcohol-aggression relationship can be explained by alcohol-induced disruption of executive cognitive functions mediated by the prefrontal cortex is critically examined. At moderate doses, alcohol has been reported to increase aggression in animals as diverse as fish, rats, cats, monkeys, and humans. Although alcohol depresses prefrontal cortex activity and disrupts executive cognitive performance in humans, alcohol's anxiolytic actions, and/or its disinhibiting effects on subcortical structures implicated in anger and aggression, may be at least as important as the higher cognitive functions cited by Dr. Giancola in accounting for the alcohol-aggression relationship. Other drugs that alter prefrontal cortex activity have also been reported to influence aggressive responding in humans and other animals, and implications of this are briefly discussed.

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In his article, Dr. Giancola attempts to integrate, under the rubric of executive cognitive functioning, a variety of higher-order functions and processes that have been implicated in the alcohol-aggression relationship. Moderate alcohol intoxication in humans has been shown to increase the likelihood of aggressive responding to provocation in a number of studies cited in the article, and is also associated with relatively selective depression of prefrontal cortex activity and temporary impairment on tasks thought to tap processes mediated by the prefrontal cortex. As examples of executive cognitive functions that can be disrupted by alcohol, Dr. Giancola cites abstract reasoning, cognitive flexibility, attention, set-shifting, planning, organization, and self-monitoring, all of which seem to require normal functioning of the prefrontal cortex.

I have long argued that many of the behavioral changes induced by alcohol in humans result from the drug's actions in the prefrontal cortex, and thus agree with Dr. Giancola's general approach to understanding alcohol's potentiating effects on human aggression. One reservation I have, however, concerns the overly cognitive emphasis of his article.

Alcohol at moderate dosage levels has been shown to increase aggressive behavior under provocation conditions not only in humans, but also in a variety of other species including fish, rats, mice, cats and monkeys (although in most animal studies, as in human studies, only some subjects were affected in this way by alcohol; Miczek, DeBold, van Erp & Tornatzky, 1997; Peeke & Figler, 1981; Yudko, Blanchard, Henrie & Blanchard, 1997). Especially in the “lower” organisms cited, an appeal to disruption of “higher” cognitive functions such as abstract reasoning, planning, and self-monitoring seems untenable, much less the four cited by Dr. Giancola as key to inhibiting an aggressive response in humans (appraisal of the situation, taking the other’s perspective, reflecting upon the possible consequences of one’s actions, and diffusing a hostile situation). Thus although disruption of such “higher” functions may very well contribute to the alcohol-aggression relationship in humans, other factors should also be considered. Anxiety, for example, can inhibit aggression, and alcohol has anxiolytic effects. Further, as Dr. Giancola notes, the prefrontal cortex is normally capable of inhibiting subcortical structures involved in the emotion of anger and its behavioral expression. If this major source of inhibitory control of anger and aggression is selectively suppressed by moderate levels of alcohol, then stimuli that are not sufficiently potent to provoke an aggressive response under normal conditions might do so when the individual is moderately intoxicated (very high doses suppress all responding). This effect might occur even when the cognitive capacities cited by Dr. Giancola remain relatively intact. As a loose analogy, consider how some patients with prefrontal lesions could correctly verbalize an appropriate strategy or response, yet were nevertheless unable to *implement* what they knew (Luria, 1964; Stuss & Benson, 1984). Similarly, in obsessive-compulsive disorder – a disease associated with abnormal activity in the prefrontal cortex - patients often realize that their compulsive behavior is inappropriate and irrational but feel an irresistible urge to engage in such actions anyway. In my view, these examples suggest that disruption of a general ability to inhibit certain kinds of emotional, habitual, reinforced, automatized, or other prepotent responding may be even more fundamental to the alcohol-aggression relationship than disruption of specifically cognitive processes per se (although the latter must often recruit the relevant inhibitory pathways in order to influence behavioral outcomes). Such a perspective is consistent with the fact that

moderate alcohol intoxication increases the likelihood of aggressive responses in a wide variety of disparate species irrespective of whether they are normally capable of the kinds of higher cognitive functions that Dr. Giancola emphasizes in his article.

A final point: if alcohol's somewhat selective depressant action on the prefrontal cortex is largely responsible for the alcohol-aggression relationship, then other drugs which affect the frontal lobes in a similar fashion should also increase aggression; conversely, drugs that activate rather than inhibit the frontal lobes should decrease aggression. These predictions are supported by evidence that, for example, such diverse drugs as cocaine and morphine also suppress frontal lobe activity (see Lyvers, 2000) and increase aggressive responses to provocation (Berman, Taylor & Marged, 1993; Davis, 1996; Long, Wilson, Sufka & Davis, 1996; Rodriguez-Arias, Minarro & Simon, 1997), whereas other equally diverse drugs that have activating effects on the prefrontal cortex, such as psilocin and other serotonergic psychedelics, MDMA or "ecstasy," and marijuana, appear to decrease the likelihood of aggression (Myerscough & Taylor, 1985; Navarro & Maldonado, 1999; Sbordone, Wingard, Gorelick & Elliott, 1979). The question can be raised, however, as to whether drugs that activate the prefrontal cortex decrease aggression by facilitating executive cognitive functioning (as Dr. Giancola's analysis might predict), or whether they do so in other ways, perhaps by their anxiogenic actions or other influences on emotional state. For example, an individual who is provoked while in the throes of a psilocin-induced psychedelic experience might become too frightened to react aggressively. On the other hand, MDMA is said to promote empathic feelings (Grilly, 1998), which would presumably inhibit aggression in a different way.

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